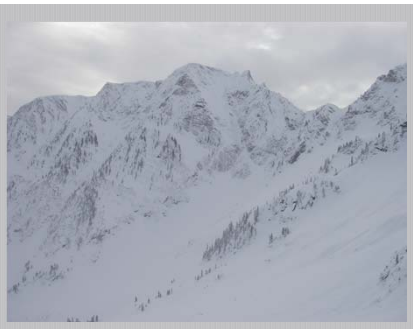
How and why would a Dentist treat OSA?

Mark A. Rasmus MD
Medical Director
Idaho Sleep Health

Co-management is key!

- Screening patients in a primary care setting
- Diagnosis of OSA
- Sleep Medicine Consultation
  - Not mandatory
  - Makes life easier!
- Treatment goals
- Treatment strategies
- Lifelong follow-up
- Continued communication

Why do we screen for OSA?



Sleep Apnea Kills!

- Wisconsin Sleep Cohort- 18yr f/u (n=1522)
  - All cause **mortality,** adjusted for age, sex, BMI was increased as severity of OSA increased
  - Adjusted Hazard Ratio 3.8 for all caused **mortality** in those w/ OSA who never used CPAP
  - Adjusted Hazard Ratio 5.2 for cardiovascular **mortality**

Young t, et al. Sleep Disordered Breathing and Mortality: Eigteen-Year Follow Up of the Wisconsin Sleep Cohort. SLEEP, 31:8, 2008.

Prevalence of OSA

- General population
  - ~4% of the general population
  - 1 in 15 has moderate to severe OSA *T Young 2004*
  - 9% women and 24% men in the middle-aged working population have OSA *T Young 1993*

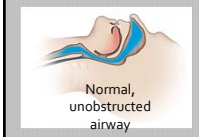
~85% not diagnosed *T Young 1997*

Defining OSA

Apnea - A cessation of airflow for ≥ 10 seconds

Hypopnea - A decrease in airflow lasting ≥ 10 seconds with a 30% reduction in airflow and with at least a 3 or 4% oxygen desaturation from baseline

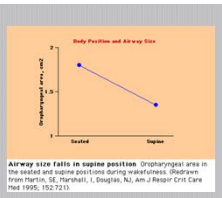
Flow limitation - Upper airway narrowing; the earliest sign of impending upper airway closure


Normal, unobstructed airway

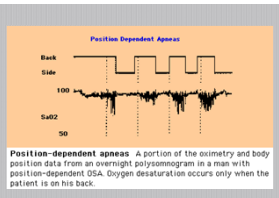

Snoring


Blocked airflow caused by obstructed airway (apnea)

Pathophysiology of OSA



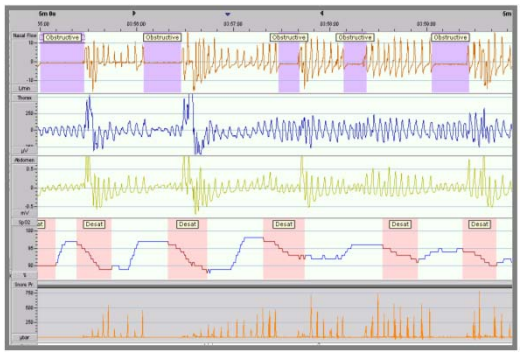
Airway size falls in supine position Oropharyngeal area in the seated and supine positions during wakefulness. (Redrawn from Martin, SE, Marshall, I, Douglas, NJ, Am J Respir Crit Care Med 1995; 152:721).



Position-dependent apneas A portion of the oximetry and body position data from an overnight polysomnogram in a man with position-dependent OSA. Oxygen desaturation occurs only when the patient is on his back.

- Oropharyngeal area decreases in the ***supine position***, which may result in position-dependent apneas

Obstructive Sleep Apnea (OSA)



ResMed

Symptoms and Risk Factors

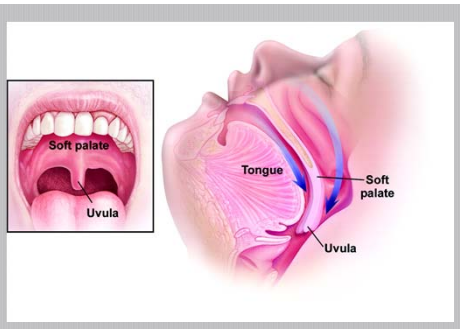
Symptoms

- Constant tiredness
- Snoring
- Poor concentration
- Depression
- Lack of energy
- Weight gain or loss
- High blood pressure

Risk Factors

- Obesity
- Male gender
- Increasing age
- Alcohol or sedative use
- Anatomic abnormalities of the upper airway
- Smoking
- Endocrine and metabolic disorders

Upper Airway Obstruction

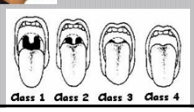


OSA Physical Exam Risk Factors

- BMI>30
- Neck circumference >16in
- High arched palate

- Micro/retrognathia

- Mallampati class airway



How do we best screen for OSA?

- History
  - Snoring w/ or w/o apnea/gasping
  - Non-restorative sleep
  - Excessive Daytime Sleepiness / Fatigue
- Co morbidities
  - Hypertension
  - GERD
  - Headaches
  - CAD/CHF
  - Cerebrovascular Dz
  - DM2
  - Metabolic Syndrome (Syndrome X)

Screening Tests for OSA

- Overnight oximetry
  - Not sensitive or specific
- STOP-BANG Questionnaire
- Polysomnography
  - Attended
  - Unattended
  - Baseline versus Split night
- Empiric CPAP

STOP-BANG Questionnaire

yes	no		
❒	❒	**Snoring:** Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?	
❒	❒	**Tiredness/fatigue:** Do you often feel tired, fatigued, or sleepy during the daytime, even after a "good" night's sleep?	
❒	❒	**Observed apnea:** Has anyone has ever observed you stop breathing during your sleep?	
❒	❒	**Pressure:** Do you have or are you being treated for high blood pressure?	
❒	❒	**Body mass index:** Do you weigh more for your height than is shown in the tables at right?	
❒	❒	**Age:** Are you older than 50 years?	
❒	❒	**Neck size:** Does your neck measure more than 15¾ inches (40 cm) around?	
❒	❒	**Gender:** Are you male?	

Scoring Rules
3 or more yes : High Risk
Less than 3 yes : Low Risk

Height	Weight (lb)	Height	Weight (lb)
4' 10"	167	5' 8"	230
4' 11"	173	5' 9"	237
5'	179	5' 10"	243
5' 1"	185	5' 11"	250
5' 2"	191	6'	258
5' 3"	197	6' 1"	265
5' 4"	204	6' 2"	272
5' 5"	210	6' 3"	279
5' 6"	216	6' 4"	287
5' 7"	223	6' 5"	295

Weights shown in the tables above correspond to BMI of 35 for a given height.

Identification of Patients with Sleep Disordered Breathing: Comparing the Four-Variable Screening Tool, STOP, STOP-Bang, and Epworth Sleepiness Scales

http://dx.doi.org/10.5664/jcsm.1308

Graciela E. Silva, Ph.D., M.P.H.[1]; Kimberly D. Vana, D.N.P.[1]; James L. Goodwin, Ph.D.[2,3]; Duane L. Sherrill, Ph.D.[3]; Stuart F. Quan, M.D.[2,4]

[1]College of Nursing – Health Innovation, Arizona State University, Phoenix, AZ; [2]Arizona Respiratory Center, College of Medicine, University of Arizona, Tucson, AZ; [3]Mel – Enid Zuckerman College of Public Health, University of Arizona, Tucson, AZ; [4]Division of Sleep Medicine, Harvard Medical School, Boston, MA

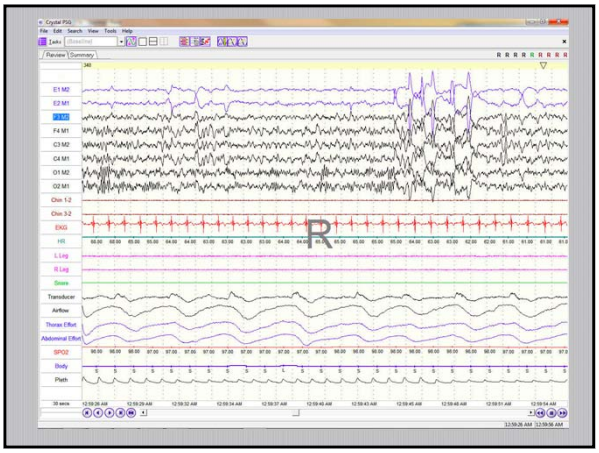
Table 2—Predictive parameters for the 4-Variable screening tool, STOP, STOP-Bang, and ESS questionnaires for moderate-to-severe SDB

	4-Variable ≥ 14	STOP	STOP-Bang	ESS ≥ 11
Sensitivity %	24.7	62.0	87.0	39.0
Specificity %	93.2	56.3	43.3	71.4
Correctly Classified %	79.4	57.5	51.0	64.8
LR+	3.7	1.4	1.5	1.4
LR–	0.80	0.67	0.30	0.85
Odds Ratio (95% CI)	4.5 (3.5–5.8)	2.1 (1.8–2.4)	5.1 (4.0–6.4)	1.6 (1.4–1.8)
Area Under the ROC (95% CI)	0.59 (0.57–0.61)	0.58 (0.56–0.61)	0.64 (0.62–0.66)	0.53 (0.52–0.56)

LR+, likelihood ratio for a positive test. LR–, likelihood ratio for a negative test. CI, confidence interval.

What do you look for during a Polysomnography?

- EEG leads (stages of sleep, arousals)
- Oximetry
- EKG
- Respiratory Effort
- Airflow
- Eye movements/ Chin myography
- Limb movements
- Capnography (in children)
- Video
- Snore microphone



Traditional Diagnostic Approach

- Attended Baseline Polysomnography
- Attended PAP Titration Polysomnography

Alternatively a Split night PSG

- Split studies may not allow enough time in supine position or REM sleep to determine adequate pressures in all circumstances
- The necessary CPAP pressure may vary considerably based upon body position and sleep stage

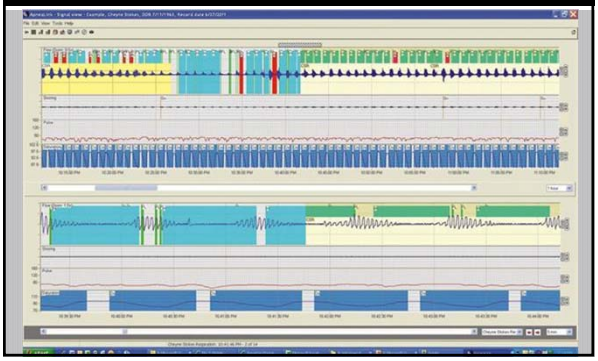
Drawbacks to Traditional PSG

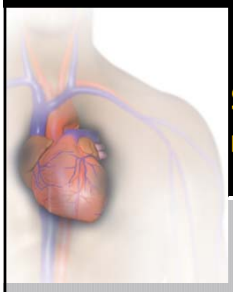
- The optimal pressure may change over time due to:
  - Weight gain or loss
  - Nasal congestion
  - Change in altitude
- Performing re-titration for recurrent symptoms is expensive, inconvenient, or delayed due to backlog in schedule

What about these new home studies?

- Unattended Polysomnography
  - Now approved by Medicare
  - Required by some insurers (BC/BS, …)
  - Not approved for those w/ comorbidities
    - Up to 90% of patients we screen!
  - No EEG waveforms
    - Sleep, awake, arousals, REM/slow wave sleep, seizures???
  - No limb movements
  - Who replaces leads when they fall off?
  - CPAP titrations???

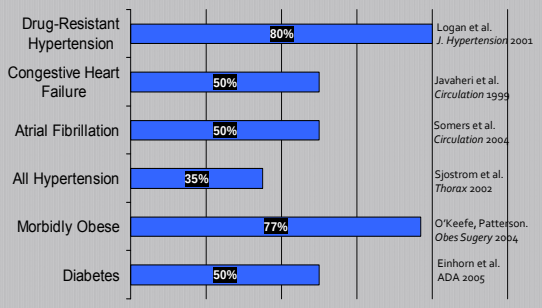
A typical ambulatory PSG





Sleep Apnea and Co-morbidities

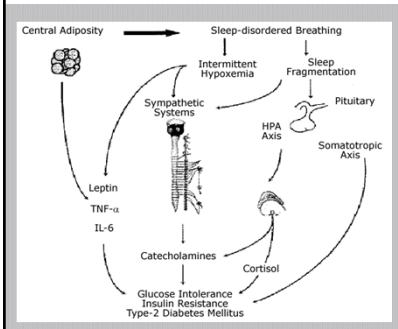
Prevalence of Sleep Apnea



Diabetes and OSA

- OSA may have a causal role in the development of diabetes
  - *(Al-Delaimy 2002, Reichmuth 2003)*
- Type 2 diabetes
  - 50% of males, 20% females *D Einhorn 2005*
  - 97% of obese diabetics *G Foster 2005*
- OSA is associated with insulin resistance (independent of obesity) *(Ip 2002, Punjabi 2002)*
- 30% of patients presenting to a sleep clinic have impaired glucose tolerance or diabetes, of which 40% are undiagnosed *(Meslier 2003)*

Possible Mechanisms

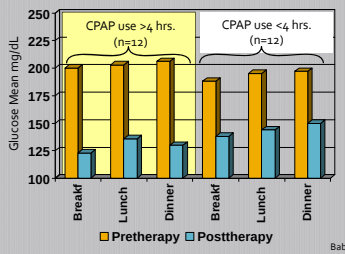


© ResMed 2006

- Increased sympathetic nervous activity
- Elevated levels of Cortisol
- Accumulation of sleep debt due to sleep fragmentation
- Recurrent intermittent hypoxia

Type 2 Diabetes, Glycemic Control and CPAP in OSA

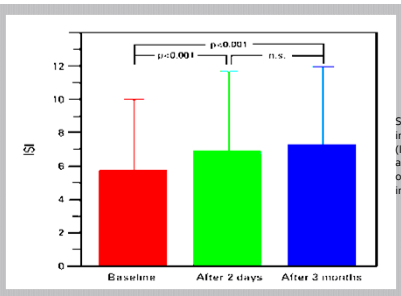
Postprandial glucose values significantly reduced with CPAP



Babue et al. *Archives of Internal Medicine* VOL 165, FEB 28, 2005

© ResMed 2006

CPAP May Improve Insulin Sensitivity



Shows improvement of insulin sensitivity index (ISI) at baseline, 2 days and 3 months after onset of CPAP treatment in 31 patients.

Harsch et al. *Am J Respir Crit Care Med.* 2004;169(2):156-162

© ResMed 2006

Conclusion

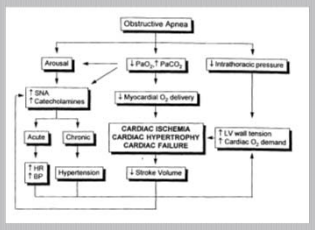
- Large population of untreated OSA sufferers
- Sleep apnea has increased in the general population
  - Aging population
  - Obesity epidemic
  - Increase in diabetes
- Serious cardiovascular consequences

Consider sleep apnea diagnosis and treatment as a tool in diabetes management

Cardiovascular Manifestations of Obstructive Sleep Apnea

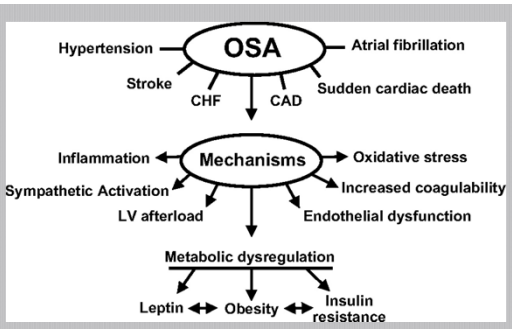
- Hypertension*
- Congestive heart failure*
- Left ventricular hypertrophy
- Diastolic dysfunction
- Pulmonary hypertension
- Stroke
- Arrhythmias
- Coronary artery disease/cardiac ischemia
- Cor pulmonale

Sleep and Cardiovascular Disease



Bradley, Journal of Cardiac Failure. 1996

Association between Cardiovascular Disease and OSA



Hypertension and Obstructive Sleep Apnea

- ~50% of patients with OSA have HTN, and ~30% of patients with HTN have OSA

Sleep Heart Health Study Conclusions

- Sleep apnea is associated with hypertension
- AHI and cumulative time with SpO2 < 90% are associated with a higher odds ratio of hypertension in a dose-response fashion
- The above findings are true after controlling for confounding variables such as age, sex, BMI, alcohol intake , and smoking

Nieto et al, *JAMA* April 12, 2000

Hypertension and OSA (other studies)

- Wisconsin Sleep Cohort Study (Peppard, et al, *NEJM*; 2000; prospective case study)
  - A relationship between OSA and HTN exists independent of age, sex, obesity, tobacco, and ETOH use
- Lavie, et al (*BMJ* 2000; retrospective population study)
  - Hypertension is associated with *apnea-hyponea index* and *nocturnal oxygen saturation nadir* in a dose-response fashion, independent of confounding variables

Hypertension and OSA

- Bixler, et al (Arch Intern Med; Aug 2000)
  - Sleep-disordered breathing, even snoring, was independently associated with hypertension in both men and women
  - Odds Ratio
    - Moderate-severe OSA = 6.85
    - Mild OSA = 2.29
    - Snoring alone = 1.56

Effect of CPAP on Hypertension

- Mayer, et al (*Cardiology* 1991)
  - After 6 months on CPAP, mean SBP 147→126 mmHg; mean DBP 82→69 mmHg
- Pepperell, et al (*Lancet* 2002)
  - Treated OSA pts with therapeutic and subtherapeutic CPAP for one month
  - Therapeutic CPAP→ ↓ mean BP 2.5 mmHg; subtherapeutic CPAP→ ↑ mean BP 0.8 mmHg

Effect of CPAP on Hypertension

- Becker, et al (*Circulation* 2003)
  - Compared therapeutic vs subtherapeutic CPAP over 9 weeks
  - Mean BP ↓ 9.9 +/- 11.4 mm Hg compared to no change with subtherapeutic CPAP
- Faccenda, et al (*AJRCCM* 2001)
  - Compared CPAP with oral placebo in normotensive subjects with OSA, and found small drop in mean BP with CPAP

Use of an Anti-Hypertensive and Odds Ratios of OSA

Age (years)	Predicted Odds Ratio	
	Male	Female
20-39	6.8	6.4
40-59	3.0	3.0
60 and above	2.1	1.4

Farney, et al; *Chest,* April 2004

How it Affects Your Practice

- Your patient with high blood pressure in a dental clinic:
  - Is the patient obese?
  - Daytime fatigue, depression, headaches?
  - Sleep-related complaints

Recommendations for Management

- Patients with hypertension should be asked if they snore, have witnessed apneas, poor sleep quality, or have excessive daytime sleepiness.
- Obese patients with hypertension should be screened for OSA
- Patients with resistant hypertension (3 meds or more to control BP) should be screened for OSA, regardless of questionnaire!

Dart, et al; *Chest* 2003

Sleep Apnea and Congestive Heart Failure

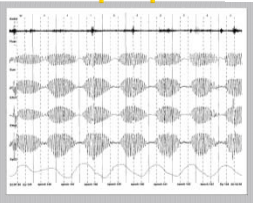
Sleep Apnea and Congestive Heart Failure

- Sleep apnea affects 2-4% of general population
- CHF affects 1.5-2% of general population
- 40-50% of stable outpatients with CHF have either obstructive sleep apnea (OSA) or Cheyne-Stokes respiration with central sleep apnea (CSR-CSA)
  - *Javaheri 1995; Naughton 1995*

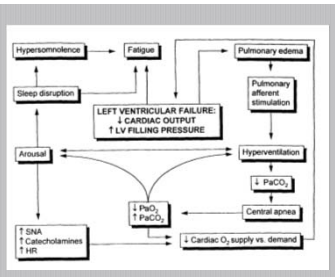
Central Sleep Apnea

- No airflow and no respiratory effort
- Pure central apnea rare, and usually caused by narcotics, anesthesia, benzodiazepines, and barbiturates
- Central sleep apnea in a **Cheyne-Stokes** pattern is seen in *CHF* and *stroke* patients
- **Central sleep apnea in a Cheyne-Stokes pattern is **a consequence** of CHF and stroke, not a cause of CHF**

CHF and Sleep Apnea



- **Cheyne-Stokes** Respiration (CSR-CSA) is a waxing and waning respiratory pattern characterized by crescendo-decrescendo breathing followed by no respiratory effort



Bradley, *Journal of Cardiac Failure, 1996*

The Research

- CSR-CSA Increases Mortality in CHF
  - At least 5 studies have shown this
    - *Andreas, et al, 1996; Findley, et al, 1985; Hanly, et al,1996; Lanfranchi, et al, 1999; Sin, et al, 2000*

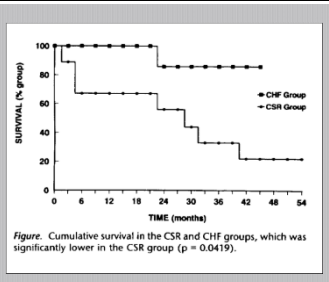
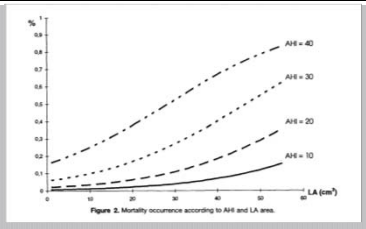


Figure. Cumulative survival in the CSR and CHF groups, which was significantly lower in the CSR group (p = 0.0419).

Hanly 1996



Lanfranchi, *Circulation 1999*

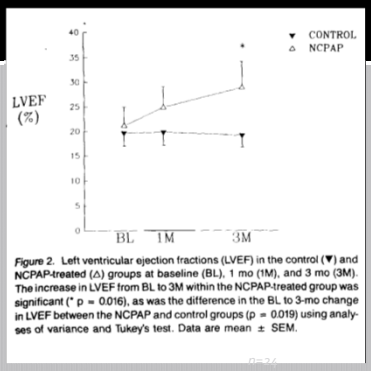


Figure 2. Left ventricular ejection fractions (LVEF) in the control (▼) and NCPAP-treated (△) groups at baseline (BL), 1 mo (1M), and 3 mo (3M). The increase in LVEF from BL to 3M within the NCPAP-treated group was significant (* p = 0.016), as was the difference in the BL to 3-mo change in LVEF between the NCPAP and control groups (p = 0.019) using analyses of variance and Tukey's test. Data are mean ± SEM.

Naughton, *Am J Respir Crit Care Med* 1995

Ejection Fraction Improves by ~7%

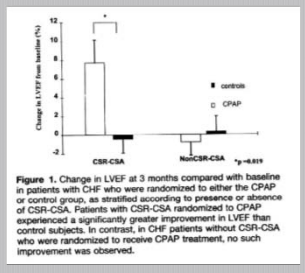


Figure 1. Change in LVEF at 3 months compared with baseline in patients with CHF who were randomized to either the CPAP or control group, as stratified according to presence or absence of CSR-CSA. Patients with CSR-CSA randomized to CPAP experienced a significantly greater improvement in LVEF than control subjects. In contrast, in CHF patients without CSR-CSA who were randomized to receive CPAP treatment, no such improvement was observed.

Sin et al, *Circulation, 2000*

Results

- Overall transplant-free survival was significantly greater in patients randomized to CPAP who complied with therapy than in control subjects

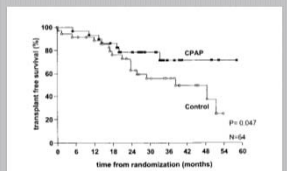


Figure 3. Treatment analysis revealed that overall transplant-free survival was significantly greater in patients randomized to CPAP who complied with therapy than in control subjects.

Sin et al, *Circulation, 2000*

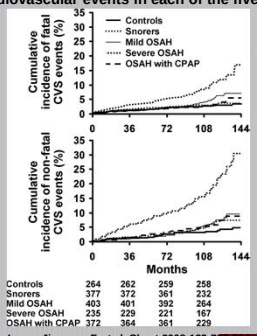
The Effect of Nasal CPAP on CHF

- Improves left ventricular ejection fraction
- Improves transplant-free survival
  - Especially if CSA/Cheyne Stokes respirations present
  - Somewhat controversial

OSA and Coronary Artery Disease

- Associated w/ AHI
- Increased CRP levels
- Hypoxemia (time and severity)
- Norepinephrine appears to be likely mediator

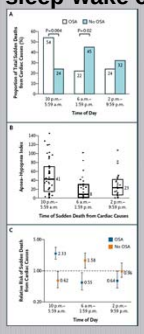
Long-term risk for cardiovascular events according to the presence of OSA and treatment with CPAP; cumulative percentage of individuals with new fatal (A) and nonfatal (B) cardiovascular events in each of the five groups studied



Lopez-Jimenez, F. et al. Chest 2008;133:793-804

CHEST The Cardiopulmonary and Critical Care Journal

Sudden death from cardiac causes according to usual sleep-wake cycles



Lopez-Jimenez, F. et al. Chest 2008;133:793-804

CHEST The Cardiopulmonary and Critical Care Journal

OSA and CVA?

- Hypoxia, Hypercapnia during UAO
- Cerebral blood flow
  - Reduced 5-28% in NREM
  - Increased 5-51% in REM
- Hypercoaguability
  - Increased blood viscosity
  - High plasma fibrinogen
  - Increased platelet aggregation
- Increased ICP during apnea

CPAP Prevents recurrent CVA

- Stroke pts w/ OSA randomized to 2 groups
  - 18 month f/u
  - CVA incidence CPAP- 6.7%
  - CVA incidence no CPAP- 36.1%
  - Odds Ratio- 5.09
- Martinez-Garcia MA. CHEST; October 2005.

Sleep apnea and Stroke/TIA

- Approximately 80% of patients w/ stroke/TIA have OSA
- Treating OSA can reduce a recurrent event by up to 50%

Other Diseases associated w/ OSA

- Headaches
  - Including Migraines
- Gastroesophageal Reflux Disease (GERD)
- Atrial Fibrillation
- Erectile Dysfunction
- Aortic Dissection
- Fibromyalgia

Questions on outcomes data?

- Hypertension
- Diabetes mellitus
- Congestive Heart Failure
- Coronary Artery Disease
- Stroke/TIA

Practice Guidelines for the Perioperative Management of Patients with Obstructive Sleep Apnea

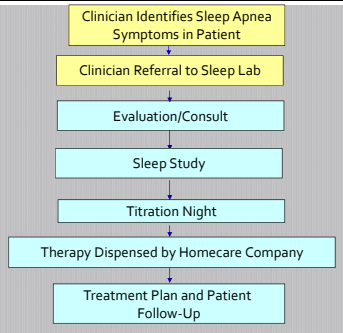
After pre-op evaluation, the anesthesiologist and surgeon should jointly decide whether to

1) manage the patient perioperatively based on clinical criteria alone

2) obtain sleep studies, a more extensive airway examination, and initiate indicated OSA treatment in advance of surgery

- 82-93% of patients with moderate to severe sleep apnea are "missed" by primary care practitioners

Diagnostic Process



How else can we work up OSA?

- Direct referral to sleep lab
- Direct referral to ambulatory PSG
  - Who interprets the study?
  - Who follows up the patient?

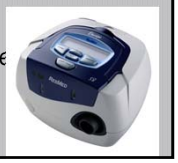
Treating Sleep Apnea

Treatment options for OSA?

- Positive Airway Pressure
  - Standard fixed pressure (CPAP)
  - bilevel
  - Autoset
- Dental appliances- "Alternative 1st line Therapy"
- Surgical options- "Second line Therapy"
  - UPPP/ septoplasty/ turbinate reduction
  - Mandibular advancement procedures

Positive Airway Pressure (PAP) is Gold Standard Treatment

- Effective in 80-90% of patients (when used appropriately)
- Therapy goals – four hours of use nightly; five nights per week
- Many comfort features available



Many Treatment Options Available

- Humidification
  - Helps prevent dry throat and nose
- Ramp
  - Helps patient fall asleep more easily
- Expiratory Pressure Relief (EPR)
- Automatic Positive Airway Pressure (APAP)
  - AutoSet™

Patient Interfaces

Full face mask for mouth breathers

Nasal cushion allows for more movement, active sleepers

Nasal pillows

Auto-CPAP (APAP)

- Different manufacturers use different variables in their algorithms
  - Snoring
  - Apnea or hypopnea
  - Airflow limitation
  - Impedance
  - Combination of above

Auto-CPAP (APAP)

- Designed to increase pressure as needed to increase airway patency
- Decrease pressure if no events detected over time

Auto-CPAP (APAP)

- Minimum and maximum pressures are set
- Mean pressure is often less than optimal fixed CPAP pressure
- May increase compliance

Auto-CPAP (APAP)

- Most units can store information regarding
  - Pressure versus time
  - Leak
  - Apnea and hypopnea information
- Such information can then be reviewed in report format

APAP Technology

- Mask or mouth leak challenging problem
  - Leaks raise baseline flow delivered by machine
  - This decreases variation in flow between inspiration and expiration
  - Interpreted as hypopnea by machine which triggers increase in flow which increases the leak

All APAP Machines Are Not Created Equally

- Different algorithms
- The algorithms are "trade secrets" and are not available for scientific scrutiny
- Cannot use term "automatic CPAP" all-inclusively since the technology is different between units

Does APAP Improve Sleep Quality and EDS?

- Yes!
- Pressure changes throughout night do not result in arousals from sleep
- Compared to placebo- APAP reduces arousals/hr, increases REM sleep, and increases SWS
- Compared to fixed CPAP- no change in sleep quality

Does APAP Improve Sleep Quality and EDS?

- APAP improves ESS (pre 16.8 vs. post 10.5)
  - Similar results to fixed CPAP
- MSLT nap latency improved from baseline (5.7 vs. 3.7 minutes $p<0.01$)
  - Similar results to fixed CPAP
- MWT nap latency improved from baseline (18.2 vs. 26.9)
  - Again same with fixed CPAP

Does APAP Prevent Hypoxemia in OSA?

- During first few minutes of REM, algorithm may be too slow to increase pressures adequately
- Most studies detect no significant difference in mean SpO2, lowest SpO2, or time spent with SpO2< 90% comparing APAP with fixed CPAP
- Concurrent oximetry recommended in initial APAP use

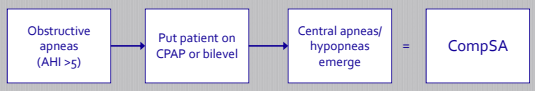
Factors to Improve CPAP tolerance

- Regular follow up
- Appropriate mask fit
- Heated humidification
- Treat nasal congestion
- Address other sleep issues
- Desensitization
- Sleep Aids / Anxiolytics

- Questions about positive airway pressure?
  - CPAP/Bipap/autoset

What is this Complex OSA that we're hearing about?

- A form of central sleep apnea
- Identified by central apneas/hypopneas emerging when treating obstructive apneas with a CPAP or bilevel device

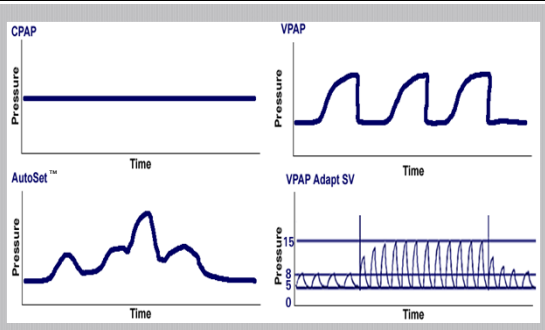


Complex OSA

- Prevalence: Estimated to be 15% of SDB population
- Not adequately treated with standard PAP treatments
  - Residual symptoms (fatigue, sleepiness, depression)
  - Intolerance to therapy
- Dramatic improvement during REM sleep (reverse of pattern seen in OSA)
- No distinguishing clinical profile

Morgenthaler et al, *Sleep*, 2006

Modes of PAP



And how does this VPAP-adapt work?

- Creates a Target Ventilation
  - The ASV algorithm monitors recent average minute ventilation (~3 min window)
  - It continuously calculates a target ventilation throughout the night (90% of recent average ventilation)
- Ventilates to the Target
  - Algorithm monitors patient ventilation and compares it to the target ventilation
  - Adjusts pressure support up or do wn as needed to achieve target

How do we treat Complex OSA? VPAP-adapt

- Initiation:
  - In the first few minutes of therapy the VPAP Adapt SV adjusts support to minimum during over-breathing and rapidly increases to maximum during apneas
  - Apneas are converted to hypopneas
  - Default settings are appropriate for most patients
- Normalized Breathing:
  - The patient's breathing is typically normalized after 10 to 40 minutes of therapy
  - For CHF patients, therapy settings should not be adjusted until after ~40 minutes to allow therapy to stabilize

How do I incorporate dental sleep medicine into my practice?

- Allow direct referrals for ambulatory or attended PSG by dentists
- Offer (and encourage!) consultations on all patients studied
- Offer MAD and/or CPAP to all patients w/ mild/mod OSA
- Encourage CPAP trial for patients w/ severe OSA
- Follow patients up indefinitely

MAD coordination

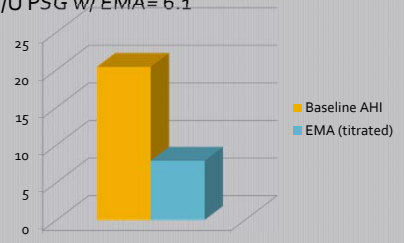
- Once fitted w/ the appliance, titration PSG is recommended!
  - Should be expected by pt prior to fitting
  - Essential to improve adherence and efficacy
  - Patients rarely know whether it's effective or not
- Combination therapy for Severe OSA patients

Oral appliance titration data

- Study of 202 consecutive patients with OSA ranging from mild to severe
- Study done using EMA appliance
- Baseline and follow-up studies all done by the same lab with the same doctor and lead technician interpreting the data

Oral appliance titration data: Overall Data

- Overall baseline AHI= 20.4
- F/U PSG w/ EMA= 6.1

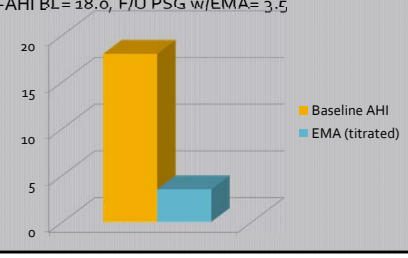


Oral appliance titration data: Responders

- Responders= AHI<10

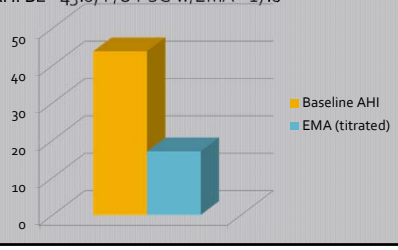
  -170/202 patients

  -AHI BL= 18.0, F/U PSG w/EMA= 3.5



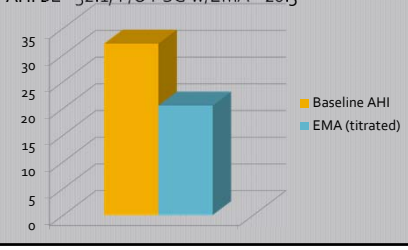
Oral appliance titration data: Partial Responders

- Partial Responders=AHI reduced by >50%
  -14/202 patients
  -AHI BL= 43.8, F/U PSG w/EMA= 17.0



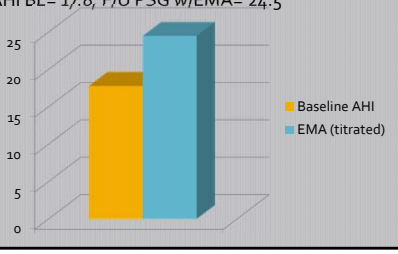
Oral appliance titration data: Poor Responders

- Poor Responders=AHI reduced by<50%
  -10/202 patients.
  -AHI BL= 32.1, F/U PSG w/EMA= 20.5



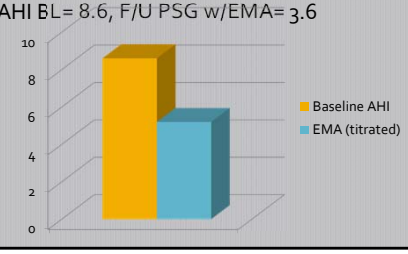
Oral appliance titration data: Non Responders

- Non Responders=AHI not reduced
  -8/202 patients
  -AHI BL= 17.8, F/U PSG w/EMA= 24.5



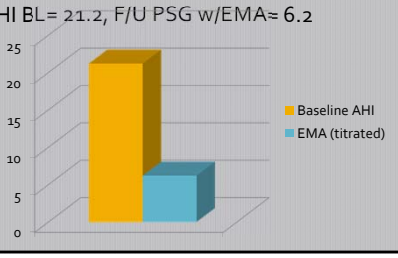
Oral appliance titration data: Mild OSA

- Mild OSA= AHI<15
  - 84/202 patients
  - AHI BL= 8.6, F/U PSG w/EMA= 3.6



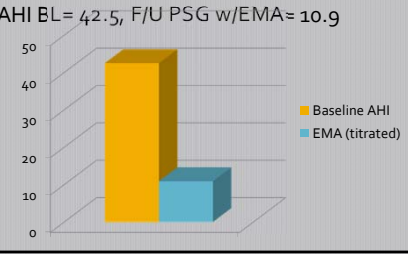
Oral appliance titration data: Moderate OSA

- Moderate OSA= AHI 15-30
  - 76/202 patients
  - AHI BL= 21.2, F/U PSG w/EMA= 6.2



Oral appliance titration data: Severe OSA

- Severe OSA= AHI>30
  - 42/202 patients
  - AHI BL= 42.5, F/U PSG w/EMA= 10.9



And WOW!

- EMA titrated to AHI <5/h in 121/202
  - 60%
- EMA titrated to AHI <1/h in 40/202
  - 20%

Why use a sleep physician?

- Medico legal issues
- We can manage comorbid sleep conditions
- Coordinate care w/ primary care and other specialty physicians
- Deal w/ the psychiatric comorbidities
- Prescribe medications/therapy/follow-up to improve adherence
- ?Better trained to see the big picture

Cost of work up?

- Attended PSG
  - Hospital ~$5000
  - Independent lab ~$2100
- Ambulatory PSG
  - ~$450

Cost of Treatment

- CPAP/APAP
  - Machine ~$2100
  - Mask ~$175
  - Renewables several times/yr
- MAD
  - Global fee ~$2500 (wide range)
- CPAP is returnable

SLEEP EDUCATION PARTNERS

SleepEdu.org

Education for the Sleep Industry

marasmus@saltzermed.com